Two cases of embolic pneumonia associated with udder cleft dermatitis in dairy cattle from the same farm

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SUMMARY

Two Holstein cows from the same farm were presented within eight months of each other with malaise, milk drop and dyspnoea. Both cows had active, or historic udder cleft dermatitis (UCD) lesions. Clinical examination and diagnostic tests led to an initial diagnosis of chronic suppurative pneumonia in both cows. The cases were poorly responsive to antibiotic treatment and died or were euthanased, 18 and 16 days respectively, after presentation. Post mortem examination of each case resulted in the diagnosis of embolic pneumonia associated with udder cleft dermatitis. In both cases UCD lesions, which were considered insignificant by the farmer and the veterinarian, are thought to have been the initiating source of infection. It is important that both farmers and vets are made aware of the possible severe consequences of udder cleft lesions and that early detection strategies for UCD and udder cleft lesions are adopted and implemented on-farm.

BACKGROUND

Udder cleft dermatitis (UCD) is a condition that affects the skin cranial to the udder on the ventral body wall. The condition has been recognised in many countries for a number of years and is sporadically seen by farmers and cattle veterinarians (Blowey and Edmonson, 1995; Warnick, 2002; Olde Riekerink; 2014; Persson Waller, 2014).

UCD can vary from mild dermatitis to an open, suppurative, foul-smelling wound which is often seemingly uncomfortable or painful on palpation and can have a long duration of up to 46 weeks, with low (5.5%) spontaneous recovery rate (Bouma et al, 2016) . It istherefore likely that this condition effects the welfare of affected cows. The area in front of the udder on the ventral body wall is hard to visualise, therefore there is a risk that UCD or other infectious or traumatic lesions

affecting this area could go unnoticed by farmers and veterinarians, until a severe or chronic infection has developed.

It has been suggested that abscessation of the milk vein secondary to infection of the adjacent skin and subcutaneous tissues could lead to rupture of this vessel and severe blood loss (Warnick et al., 2002), however UCD lesions are generally not considered to cause systemic illness in themselves, although they have been reported to increase the risk of clinical mastitis (Persson Waller et al, 2014). This case report details two cases from the same farm in which systemic illness and subsequent death, or euthanasia for welfare reasons, occurred secondary to a septic thrombus from lesions of the skin cranial to the udder. In both cases the UCD lesions had been considered insignificant or the extent of the infection had gone unnoticed by both the farmer and the veterinarian.

CASE PRESENTATION

The two cases described below occurred on the same dairy farm in February (Cow 1) and September (Cow 2), 2016. The farm is a client of Langford farm animal practice, a first-opinion practice run by the University of Bristol and both cases were managed by the farms regular first-opinion vet.

The herd comprises 200 Holstein cows, calving all year round. Cows are milked three times daily with an average 305-day yield of 10,500 L. Milking cows are run as two groups according to milk yield, and both groups are housed all year round in sand-bedded cubicles. The herd is endemically infected with both digital dermatitis and interdigital necrobacillosis. All milking cows are foot-bathed twice weekly in 3% formalin to control these diseases. Digital dermatitis scoring during December 2016 indicated a herd prevalence of 33% with 10% active lesions, equivalent to score M2 of the M-scoring system (Dopfer et al., 1997, Greenough. P.R., 2008). Foot health is otherwise good with prevalence of lameness ranging from 5–15% in 2016.Udder cleft dermatitis lesions are recognised and treated by the farm staff and while the actual prevalence was unknown at the time either case was presented, farm staff have subsequently reported the prevalence to be approximately 7% (15 affected cows at any one time) with approximately 2% (4 cows) having ‘significant’ lesions that are treated by the farm staff.

Ongoing bulk-milk tank testing suggests that the herd is free of IBR and there had been no reported incidence of contagious respiratory disease in the adult herd in the weeks or months preceding the presentation of the cases.

Both cows described here were pedigree adult Holsteins, in mid-lactation (127 and 204 days, respectively) with a projected 305-day yield of 10,500 L for their current lactation.

Cow 1

A 6.5-year-old pedigree Holstein cow in 4th lactation was presented with milk drop, dyspnoea, inappetence and malaise.

Cow 2

A 3.5-year-old 2nd lactation Holstein cow lactation was presented with malaise, milk drop and dyspnoea.

INVESTIGATIONS

Investigations (Cow 1)

On initial clinical examination the cow was dyspnoeic (respiratory rate 60 breaths per min) with increased lung noises over the cranioventral lung fields, particularly over the left lung field. Cardiac auscultation revealed a slightly increased heart rate (90 beats per minute) but cardiac rhythm and sounds were normal. The cow had poor rumen fill (2/5) and decreased rumen turnover. She was dehydrated, and dry faeces was found in the rectum upon rectal examination. The cow was non-pyrexic, and clinical examination revealed no other significant findings.

On subsequent examination two days later, the cow had a swollen right udder fore-quarter from which purulent, clotted milk was expressed as well as enlarged supramammary and precrural lymph nodes.

Eleven days after initial presentation, milk from the right fore-quarter was normal, but the cow was still dyspnoeic with increased lung sounds in the cranioventral lung fields.

At initial examination a sample was submitted for haematological examination. Complete blood count and differential counts showed a mild haemoglobinaemia, neutrophilia including the presence of band neutrophils and a lymphopenia. Platelets were reported to be plentiful on a blood smear (Figure 1)

Initial problem list

Dyspnoea

Inappetence

Decreased rumen fill and decreased rumen turnover

Malaise

Increased cranioventral lung noises

Mastitis

Neutrophilia

Lymphopenia

Treatment

After initial assessment a presumptive diagnosis of chronic, suppurative bacterial pneumonia was made. The cow was treated with meloxicam (0.5mg/kg; Recocam, Bimeda, UK) and amoxicillin with clavulanic acid (8.75 mg/kg; Combiclav, Norbrook, UK). Oral rehydration therapy was also administered (20L water with rehydration salts; Selekt Restore, Nimrod, UK).

Two days after initial assessment when the cow was presented a second time, a diagnosis of clinical mastitis of the right fore-quarter was also made. At this time, a repeat dose of meloxicam (0.5mg/kg; Recocam, Bimeda, UK) was administered, and instruction was given to the farmer to continue treatment with amoxicillin and clavulanic acid until the clinical mastitis had resolved. Oral rehydration therapy was repeated, and the farmer was advised to strip the affected quarter out at least twice daily until the milk appeared normal.

Sixteen days after initial presentation the cow was presented as being weak with severe bleeding bilaterally from the nostrils. At this time the decision was made to euthanase the cow for welfare reasons, and the carcass was submitted to the University of Bristol Pathology department for post-mortem examination.

Post mortem examination

At post-mortem examination a small (5cmx10cm) crusted lesion as well as multiple irregular nodular swellings were found on the abdominal skin. The nodular swellings extended from the cranial udder to the mid-abdomen. These swellings were abscesses, with brown foul-smelling fluid and some caseous material present. The right precrural lymph node was similarly abscessed. The supramammary lymph nodes were also enlarged, and abscessation grossly similar to that found in the skin (dark brown purulent liquid) was observed in the right fore-quarter of the udder (Figure 2).

The liver was enlarged and adhered to the diaphragm in many places. The rumen content was dry, and large blood clots were present dorsally in the rumen. The pelvic lymph nodes were enlarged.

Examination of the cardiovascular system revealed a firm, 1cm yellow endocarditis lesion on the pulmonary semi-lunar valve closest to the interventricular septum.

Examination of the lungs revealed multiple abscesses within much of the caudal left lobe and a large blood clot in the centre of the lobe, as well as dark, foul-smelling purulent liquid, similar to that seen in the skin and udder (Figure 3). The pleura were extensively adhered to the thoracic wall. Smaller areas of similar, well-encapsulated abscessation were found throughout the left lung lobes as well as the right caudal lobe, containing varying amounts of dark purulent material. The kidneys were markedly enlarged.

Bacterial culture

Routine culture on MacConkey and blood agar plates at 37° C for 24 hours was performed (anaerobic cultures were not performed at this time for economic reasons); pure growths of a mucoid non-lactose fermenting organism were isolated from the skin and udder. The same organism was isolated as the predominant organism in a mixed growth (also containing Mucor spp) from the lungs. This pathogen was identified as Providencia stuartii.

Investigations (Cow 2)

Clinical examination

Upon initial clinical examination the cow had increased heart rate and respiratory rate as well as increased respiratory effort. She had increased lung sounds in the ventral lung fields, more evident on the left hand side. At this time the veterinarian was made aware of dermatitis of the skin cranial to the cow's udder which was being washed with a hose in the parlour twice daily and sprayed with oxytetracycline spray (Engemycin spray 25mg/ml, MSD). The cow was non-pyrexic and no other abnormalities were found.

At initial presentation, a blood sample was submitted for haematological examination. The complete blood count revealed a mild normocytic, normochromic anaemia. A neutrophilia, including the presence of neutrophils with cytoplasmic basophilia (toxic effects), and lymphopenia were also present. Platelets were reported to be plentiful on a blood smear (Figure 1).

A diagnosis of bacterial pneumonia was made, and treatment was initiated.

Seven days later the cow was re-examined including bilateral ultrasonography of the lung fields. Ultrasonographic examination revealed areas of hyperechoic irregularity in the ventral lung fields consistent with the presence of consolidated lung (Scott, 2013). The left lung fields were more severely affected. These findings were considered to support the initial diagnosis of pneumonia.

Initial problem list

Milk drop

Dyspnoea

Lung consolidation

UCD

Anaemia

Neutrophilia with toxic effects

Lymphopenia

Treatment

At initial presentation the cow was treated with meloxicam (0.5mg/kg; Recocam, Bimeda, UK) and amoxicillin with clavulanic acid (8.75 mg/kg; Combiclav, Norbrook, UK). Oral rehydration therapy was administered (20 L water with rehydration salts; Selekt Restore, Nimrod, UK).

After seven days the cow was re-assessed, and the farmer requested that if the cow were to stay on antibiotic treatment any longer the treatment be changed to a less expensive antibiotic. The cow was subsequently treated with amoxicillin (7mg/kg; Duphamox, Zoetis, UK) for a further 11 days.

Eighteen days after initial presentation the cow was found dead in the cubicles and was submitted for gross post-mortem only, at the University of Bristol Pathology department.

Post-mortem examination

Post-mortem examination revealed a large ulcerated lesion (20 x 15 cm) cranial to the udder (Figure 4). The ulcerated lesion was bordered by an area of fibrosis and thickened cutaneous and subcutaneous tissue including multiple abscesses containing dark brown material. This area of inflammation and infection extended laterally, surrounding the left milk vein. A large thrombus was found in the milk vein which extended forwards towards the thorax (Figure 5). The precrural lymph nodes were enlarged bilaterally.

The liver was enlarged with rounded borders. Numerous small (1-2 cm) ulcers were found over the gastric and pyloric mucosa of the abomasum. Cardiovascular examination revealed no abnormal findings.

Pleural adhesions were present between the left cranial lung lobe and the thorax. Several small abscesses were found in the left cranial lung lobe (ranging from 2-4 cm), and there was marked emphysema throughout the lobe. The ventral aspect of the right cranial lung lobe was consolidated and dark red. A larger abscess (20 cm diameter) was also found in the right cranial lobe which contained malodorous dark fluid (Figure 6). Extensive emphysema was seen throughout the right lung and the mediastinal lymph nodes were enlarged.

No further diagnostics were performed as, for economic reasons, the farm only elected a gross post-mortem. Although not confirmed by culture, it was noted that the abscesses found throughout the carcass of this cow all had a similar appearance, containing dark, malodorous fluid, so it is likely that common pathogens (e.g. Fusobacterium necrophorum, Trueperella pyogenes, or other opportunistic pathogens) were involved.

Cause of death was considered to be septicaemia.

DIFFERENTIAL DIAGNOSIS

While it is assumed that the site of the initial infection in both of the cases presented above was the lesion on the ventral abdomen, cranial to the udder, it should be considered that there could have been a different site of initial infection. As well as the skin lesion and nodular swellings in the skin cranial to the udder, Cow 1 also had abscesses in the udder, one precrural lymph node and the lungs as well as a lesion on the pulmonary semi-lunar valve. Whilst it may be possible that the udder was the initial site of infection, given the extent of the dissemination, it is likely that a chronic infectious process occurred. The cow had not had any previous cases of mastitis in the current lactation and had a consistently low somatic cell count recorded, suggesting that the udder was less likely to be the initial site of infection. The precrural lymph nodes are responsible for drainage of the caudal

abdominal wall and are not strictly drainage lymph nodes of the udder (Divers, 2007); therefore abscessation of this node is more likely to be a result of infection of the abdominal wall, whereas an intramammary infection would have been more likely to have been drained by the supramammary lymph nodes. While enlarged, no abscesses were found in the supramammary lymph nodes.

Multiple small abscesses, rather than a single, solitary abscess, were found throughout the lungs of both cows. This diffuse abscessation is indicative of haematogenous bacterial spread via a septic embolus from another site, as opposed to a primary pneumonia (Zachary, 2016). Furthermore, as a facultative anaerobic bacteria, the presence of Providencia stuartii in the lung suggests its origin to be an alternative site. The presence of a valvular endocarditis lesion in cow 1 also supports the theory of haematogenous bacterial spread in this animal. It is therefore likely that the initial site of infection in both of the cows was the abscessed ventral body wall, cranial to the udder, rather than a lung abscess.

Thrombophlebitis of the milk vein (Cow 2) would more commonly be a consequence of non-sterile venipuncture of this vein, however this is not common practice on this farm. It was confirmed that neither the veterinarians nor the farm staff had injected into this site in this cow.

The liver enlargement was considered to be secondary to the septicaemia, however the adhesions between the liver and the diaphragm were not considered to be associated with the UCD lesion or its embolic spread, and were thought likely to be secondary to an historic insult affecting the liver eg; Fasciola Hepatica infection.

Clinical presentation of a cow with severe bilateral epistaxis (Cow 1) is more commonly associated with end-stage presentation of chronic subacute ruminal acidosis (SARA) following pulmonary emboli secondary to hepatic abscessation. Post-mortem findings associated with ongoing SARA may also include rumenitis (Kleen et al., 2003). Neither rumenitis nor hepatic abscessation were observed on post-mortem examination of either cow. Furthermore, gross inspection of the ruminal epithelium, as described by Rezac and colleagues (Rezac et al., 2014), did not indicate acidosis to be a concern in this cow.

TREATMENT

See ‘Investigations’ section.

OUTCOME AND FOLLOW-UP

The findings of the post-mortem examinations and the culture results from Cow 1 were reported to the farm manager and stockman. After the findings of the first case were reported, a conversation was initiated with the farm manager and stockman as to whether the swellings and abscessation on the cow's ventral abdomen were secondary to a traumatic injury or infectious cause. At this time the stockman reported treating the cow for an UCD lesion, which he had considered to have healed at least two weeks before the cow was noticed to be ill. At this time UCD was not considered to be the cause of the deep skin abscesses. The cubicles, loafing areas, cow passageways and milking parlour were searched for any protruding objects that could have caused injury. The sand used for bedding was also inspected for sharp objects and glass; none were found, so no definitive cause was agreed upon.

After the post-mortem results for Cow 2 were reported, it was considered more likely that UCD had in fact played a role as the initiating cause of the subsequent bacterial dissemination to the udder, lymph nodes and lungs of Cow 1. The UCD lesion in Cow 2 was considered to be the primary site of infection, leading to infection and thrombosis of the adjacent milk vein and subsequent emphysema and septic embolus to the lungs.

Since the cause of the pathology in both cases was considered to be an UCD lesion or other infection cranial to the udder resulting in deep cutaneous, subcutaneous and venous infiltration, the herdsman was made more aware of the importance of checking for these lesions during milking in the parlour and treating mild infections promptly. The externally contracted foot-trimmer was also informed of the concern regarding these lesions and will now inform the stockman if he observes them on cows whilst performing routine foot trimming. This takes place in a hydraulic tipping crush, thus allowing full visualisation of the abdomen cranial to the udder.

A protocol for treatment of these lesions has also been drawn up which involves assessment of any known UCD lesion or infection cranial to the udder by the stockman. Lesions that do not involve broken skin or only crusting should be cleaned and debrided daily and sprayed with topical antibiotic spray (Engemycin spray, MSD, UK) after each milking to avoid the risk of contamination of milk. Lesions that have open wounds or exudate such as pus or blood should be assessed by a vet and considered for a course of systemic antimicrobials, ideally following culture and sensitivity.

DISCUSSION

The two cases described in this case report illustrate how infection or trauma in the area cranial to the udder on the ventral body wall can progress to a deep infection of the skin and subcutaneous tissues, the full extent of which can easily go unnoticed and subsequently untreated by both farmers and veterinarians.

While in the second case an active UCD lesion was present (score 4 in the scoring system devised by Olde Riekerkink et al, 2014), the first case did not have any open wounds or sites of active infection on the ventral abdomen (score 3, Olde Riekerkink et al, 2014).

At post mortem of the second case the full extent of the infection became evident as multiple swollen areas containing dark, foul smelling purulent and caeseous material were found cranial to

the udder on the ventral body wall. Although the origin of this infection cannot definitively be associated with a previous UCD lesion this is considered to be the most likely cause of initial infection in this area due to the fact that no other causal factor could be identified and no other cows in the herd have been observed to have wounds to their ventral abdomen or udder, either before or since the presentation of this case.

The causal agents involved in UCD are a topic of debate and a definitive causal agent has not been identified. It has been suggested that UCD lesions are caused by the same pathogens that cause bovine digital dermatitis (BDD)(Boyer and Singleton, 1998), although other studies have not found BDD to be a risk factor for UCD (Warnick et al., 2002). It is likely that if BDD-causing treponemes are involved, they are not the sole pathogens associated with these lesions (Evans et al., 2010). Further studies into this area have identified a wide variety of genetic diversity within the treponeme species found in UCD lesions (Stamm et al., 2009). Silver staining for treponemes was not carried out in either case presented above.

The bacteria cultured from multiple lesions from the first cow, Providentia stuartii, is a coliform bacteria, usually found in soil, water and sewage and is generally considered to be an opportunistic pathogen. Although Providentia stuartii was cultured from the skin, udder abscess and lungs of the first case, indicating its involvement in the formation of abscesses in these three places, it cannot be presumed that this was the causal organism, rather it is considered to be an opportunistic pathogen which caused a secondary infection after an initial insult, be it trauma or UCD infection. Furthermore, the unusual gross appearance of the abscesses (dark brown / black fluid with a foul smell) in both cases suggest a similar initiating factor and/or pathogen involvement, one that is likely to include anaerobic bacteria. Trueperella pyogenes is likely to be present, as has been the case with similar cases submitted later to the pathology service from other farms (Millar et al. 2017). Fusobacterium necrophorum has also been isolated in these subsequent cases, although this was not found in another study (Evans et al., 2000) which did isolate T.pyogenes alongside many other bacterial species.

Despite the fact that the farmer was aware of the infection cranial to the udder in both cases they had been managed using only topically applied antibiotics and washes, and veterinary advice had not been sought with regard to the treatment of these lesions on either occasion. It is likely that these lesions commonly go unreported or examined by vets. Wounds or infections of the skin cranial to the udder including UCD, are not thought by farmers to be a common problem, but UCD is considered to be an important health problem (Persson Waller et al., 2014). Farmers are concerned about these lesions due to the pain they cause the cow, they are foul smelling, they may be associated with mastitis, and there is no effective treatment for them. The fact that no one treatment (including the use of systemic antibiotics) is considered to be consistently effective is also supported in the findings of a study in Dutch dairy cattle (Amersfort, 2012). This lack of satisfactory resolution of lesions after treatment, the fact that the majority of farmers questioned in one study reported not to consider UCD lesions to be an important health problem (Persson Waller et al., 2014), and the slow progression of the lesions (Bouma et al, 2016) may result in farmers under-reporting these lesions to vets and initiating treatment without veterinary consultation.

The post mortem findings of both of the cases presented in this case report suggest that what was considered to be a superficial infection by both the farmer and the vet had in actual fact become a deep infection of the subcutaneous tissues of the ventral abdomen cranial to the udder. In cow 1 the lesion cranial to the udder that had undergone topical treatment by the farmer and had appeared to resolve, had remained within the deeper tissues as areas of abscessation and fibrosis. In

both cases the infection had then disseminated to other tissues including haematogenous spread to the lungs.

To the authors knowledge this is the first report of UCD or infection cranial to the udder that has resulted in haematogenous spread of infection and resulted in the subsequent death of the affected animals. The fact that such aetiology has not been reported previously may be because farmers and vets have not considered UCD or infection of the udder cleft to cause deeper infection and so if cows were presented with udder cleft lesions and concurrent respiratory disease, the two conditions were not thought to be associated. It could also be that, as in cow 1, a previously active infection had healed to some extent and was no longer obvious to either the farmer or the vet as an active infection, despite chronic infection remaining in the subcutaneous tissues. Furthermore, if lung, or other systemic abscessation was found at post mortem examination, the lesions in the abdominal wall may not have been noticed, or thought to have been associated with the abscesses seen in the lungs.

UCD and other udder cleft lesions are not consistently reported to vets before treatment is initiated and therefore the majority are treated empirically, rather than as the result of culture and sensitivity testing. Without an understanding of a definitive causal organism, defining an appropriate empirical treatment protocol for udder cleft lesions is difficult. While many treatment options, both topical and systemic have been reported, none have been found to be more successful than others (Amersfort, 2012). As well as the increased treatment costs and potential economic losses that are associated with the use of systemic antibiotics, both farmers and vets should be considerate of ever-increasing public and market pressures to reduce antimicrobial use in dairy cattle. Indeed, the use of systemic antimicrobials is hard to justify in order to treat a condition which is generally considered to cause only a superficial infection and which is not associated with production losses (Persson Waller et al., 2013). Even the use of topical antimicrobials in these cases should continue to be reviewed as non-antibiotic treatments (eg: honey infused bandaging) have been found to be beneficial in wound management in bovines (Tomlinson, 2016).

In the light of the limited effectiveness of treatment options, reducing the risk of the development of these lesions should be considered. Risk factors for UCD including udder conformation (large front quarters and small angle between udder and abdominal wall) (Olde Riekerkink et al., 2014, Persson Waller et al., 2014), breed (Swedish red cows were found to be at higher risk), high milk yield and parity (3 or more lactations) have been identified (Persson Waller et al., 2014). Other factors including endemic digital dermatitis infection, the use of cow cubicles (Boyer and Singleton, 1998) and the use of footbaths (Olde Riekerkink et al., 2014) have been suggested. Given that the risk factors identified (breed, high milk yield and greater parity cows) may be both difficult to change and associated with more profitable animals on a farm, the economic benefits of reducing these risks are unknown. Furthermore, the incidence of systemic illness and cow losses due to the progression of disease as described in this case report is currently unknown. It is therefore important that both farmers and vets adopt strategies to allow early detection of these lesions so that treatment can be initiated as early as possible and monitored until full resolution.

LEARNING POINTS/TAKE HOME MESSAGES

- Infection at the junction of the cranial border of the udder and the body wall, whether caused by injury and secondary infection or primary UCD can progress to chronic infection of the subcutaneous tissues and can then disseminate both via lymphatic drainage or haematogenously to other sites, including the lungs.

- Widespread dissemination of infection from these lesions can lead to sepsis and subsequent death or rupture of pulmonary vessels and severe haemoptysis.

- It is important that both farmers and vets are made aware of the possible severe consequences of udder cleft lesions that invade deep into the tissues of the ventral abdomen of cows.

- It is crucial that early detection strategies for UCD and udder cleft lesions are adopted and implemented on-farm and that farmers present severe or non-healing cases, particularly those with local oedema or swelling around the lesions, to vets for further investigation, treatment and follow-up.

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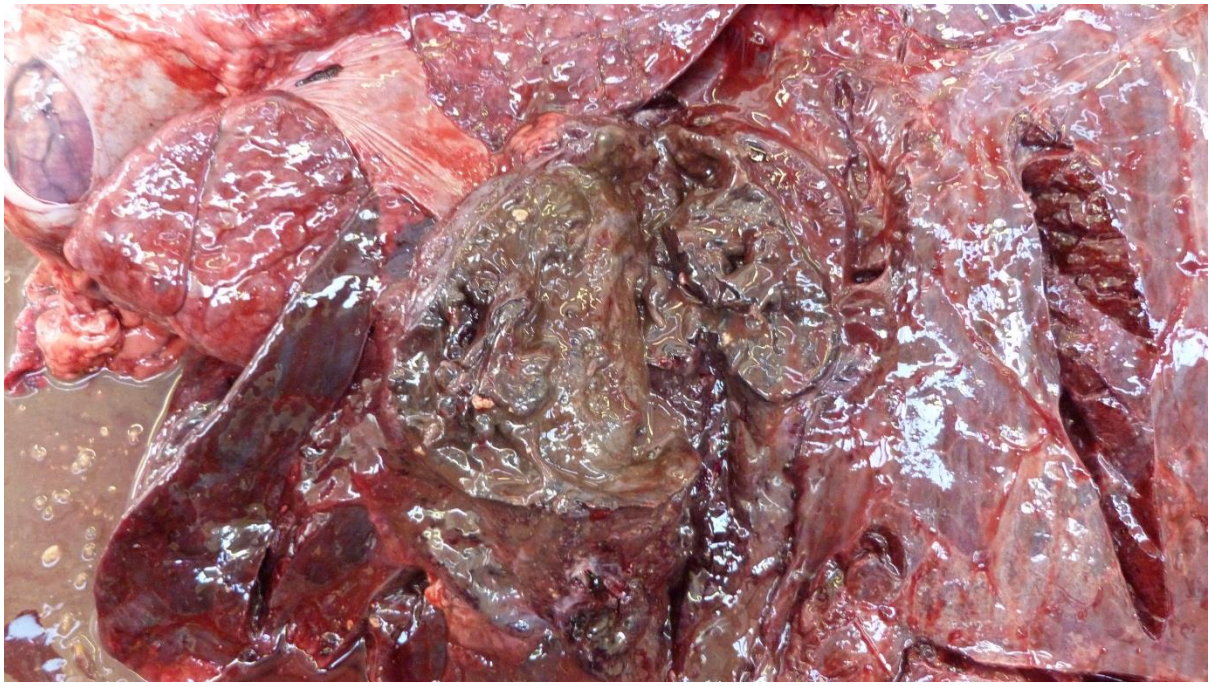
	Cow 1	Cow 2	Reference interval
Haemoglobin (g/dl)	12.6	10.0	9.0 – 12.0
Haematocrit (%)	37.4	26.2	25.8 – 40.0
Red blood cells ($x10^{12}$/L)	6.9	5.4	5.2 – 9.2
Mean cell volume (fl)	54.0	48.0	44 - 60
Mean cell haemoglobin concentration (g/dl)	34.7	38.1	34 – 38
Platelets ($x10^9$/L)	112	388	412 - 1003
White blood cells ($x10^9$/L)	9.81	9.58	6.2 – 13.6
Band Neutrophils ($x10^9$/L)	0.49	Not reported	0.0 – 0.3
Neutrophils ($x10^9$/L)	6.77	6.77	1.1 – 3.6
Lymphocytes ($x10^9$/L)	1.47	2.09	4.0 – 9.8
Monocytes ($x10^9$/L)	1.08	0.47	0.2 – 1.3
Eosinophils ($x10^9$/L)	0.00	0.10	0.0 – 0.7
Basophils ($x10^9$/L)	0.00	0.08	0.0 – 0.3



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Mean cell volume (fl)	54.0	48.0	44 - 60
Mean cell haemoglobin concentration (g/dl)	34.7	38.1	34 – 38
Platelets ($x10^9$/L)	112	388	412 - 1003
White blood cells ($x10^9$/L)	9.81	9.58	6.2 – 13.6
Band Neutrophils ($x10^9$/L)	0.49	Not reported	0.0 – 0.3
Neutrophils ($x10^9$/L)	6.77	6.77	1.1 – 3.6
Lymphocytes ($x10^9$/L)	1.47	2.09	4.0 – 9.8
Monocytes ($x10^9$/L)	1.08	0.47	0.2 – 1.3
Eosinophils ($x10^9$/L)	0.00	0.10	0.0 – 0.7
Basophils ($x10^9$/L)	0.00	0.08	0.0 – 0.3